Broccoli, cauliflower and genetic cancer

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Need another reason to eat vegetables? A new study at Rutgers shows that certain vegetables – broccoli and cauliflower, in particular – have natural ingredients that may reduce the risk of developing hereditary cancers.

A research team led by Rutgers' Ah-Ng Tony Kong has revealed that these widely consumed cruciferous vegetables – so called because their four-petal flowers resemble crosses – are abundant in sulforaphane (SFN). This compound had previously been shown to inhibit some cancers in rodents induced by carcinogens – substances or agents external to the body. Kong's investigations, however, focused on whether SFN might inhibit the occurrence of hereditary cancers – those arising from one's genetic makeup.

The American Cancer Society estimates that more than two-thirds of cancer may be prevented through lifestyle modification, and nearly one-third of these cancer occurrences can be attributed to diet alone.

"Our research has substantiated the connection between diet and cancer prevention, and it is now clear that the expression of cancer-related genes can be influenced by chemopreventive compounds in the things we eat," said Kong, a professor of pharmaceutics in the Ernest Mario School of Pharmacy at Rutgers, The State University of New Jersey.

Chemopreventive properties are those that prevent, stop or reverse the development of cancer. In a study published online in the journal Carcinogenesis, Kong and his colleagues used a mouse model for human

colon cancer to demonstrate the chemopreventive power of SFN and explain how it works to thwart cancer at the biomolecular level.

The researchers employed a specially bred strain of mice (labeled Apc/Min/+) that carry a mutation that switches off a gene (Apc) that suppresses tumors. This is the same gene known to be directly implicated in the development of most colon cancers in humans. When the gene is inactivated in the mice, polyps, which lead to tumors, appear spontaneously in the small intestine. Experiments using these mice can help in designing human clinical trials that can lead to new treatments for colon cancer in humans.

Two groups of mice were fed diets supplemented with SFN for three weeks, one group receiving 300 parts per million (ppm) of SFN and the other getting 600 ppm. "Our results clearly demonstrated that those mice fed with an SFN-supplemented diet developed significantly fewer and smaller tumors," Kong said.

After the three weeks, the average number of polyps in the small intestine in each mouse decreased more than 25 percent in those on the 300 ppm diet and 47 percent in the 600 ppm treatment group, as compared to control animals who had received no SFN.

"Our results showed that SFN produced its cancer preventive effects in the mice by inducing apoptosis (programmed cell death) and inhibiting proliferation of the tumors; however, it was not clear what mechanism SFN employs to accomplish this," Kong said.

Using biomarkers (indicator molecules) associated with apoptosis and proliferation, Kong's team found that SFN suppressed certain enzymes or kinases that are highly expressed both in the mice and in patients with colon cancer. The researchers concluded that this enzymatic suppression activity is the likely basis for the chemopreventive effects of SFN.

"Our study corroborates the notion that SFN has chemopreventive activity. Based on these findings, we feel SFN should be evaluated clinically for its chemopreventive potential in human patients with Apc related colon cancers," Kong said.

Source: Rutgers, the State University of New Jersey